86 Abdominal Pain

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Definition

Pain in the abdomen is the single most important symptom of an acute abdominal pathologic process. It is the symptom that brings the patient to his physician and the symptom that deserves the utmost care in evaluation. It has been said that a skilled clinician can identify the source of abdominal pain from the history alone 80 to 90% of the time. To achieve that goal requires a thorough understanding of the pathogenesis of the many abdominal diseases that produce pain, and the pathways over which it is transmitted.

Technique

It is imperative to analyze abdominal pain with particular emphasis on six features: onset, progression, migration, character, intensity, and localization.

Onset

Abdominal pain may be of sudden, rapid, or gradual onset. Pain of sudden onset occurs within a second. The patient will relate the time of onset at a precise moment, usually stating exactly what activity was going on at the time the pain began. Sudden onset of pain is commonly associated with perforation of the gastrointestinal tract from a gastric or duodenal ulcer, a colonic diverticulum, or a foreign body. Other common causes include a ruptured ectopic pregnancy, mesenteric infarction, ruptured aortic aneurysm, and embolism of an abdominal vessel.

Pain of rapid onset begins with a few seconds and steadily increases in severity over the next several minutes. The patient will recall the time of onset in general but without the precision noted in pain of sudden onset. Pain of rapid onset is associated with cholecystitis, pancreatitis, intestinal obstruction, diverticulitis, appendicitis, ureteral stone, and penetrating gastric or duodenal ulcer.

Pain of gradual onset is pain that slowly becomes more severe only after a number of hours or even days have elapsed. The patient's memory as to the time of onset of the pain is vague; he or she can pinpoint only the day or possibly the week of onset. Pain of gradual onset is commonly associated with neoplasms, chronic inflammatory processes, and large bowel obstruction. Many other intra-abdominal conditions are associated with pain of gradual onset, making an accurate diagnosis from the history more difficult with this symptom than in pain of sudden or rapid onset.

Progression

It is of real diagnostic significance to determine the progression of the pain over the interval from the time of onset until the patient seeks medical attention. Has the pain abated, or has it increased? Have there been intervals of total absence of the pain, or has the pain always been present, changing only in character? From this information the clinician may be able, for example, to identify a perforated ulcer that has produced pain of sudden onset that subsequently abates dramatically if the perforation seals and no further leak occurs.

Intermittent attacks of abdominal pain that progress to a steady, constant ache suggest a small bowel obstruction with vascular compromise that will lead to the grave complication of necrosis of the involved incarcerated bowel.

Migration

Pain that "shifts" from the original site of onset to another location in the abdomen is most often associated with acute appendicitis where periumbilical or epigastric pain (visceral) that is present early in the course of the disease is replaced with right lower quadrant (somatic) pain later in the illness when the parietal peritoneum becomes involved with the inflammatory process.

Pain produced by irritation of the parietal peritoneum by duodenal contents leaking from a perforated duodenal ulcer may begin in the epigastrum and may migrate to the lower quadrants of the abdomen or pelvis depending on the pathway that the leaking material takes through the abdominal cavity.

Character

Determining the character, or what kind of pain the patient is experiencing, is of prime importance in discovering the pathologic process responsible for it. Since the patient's description of the pain must be purely subjective, it is essential that the clinician and the patient communicate clearly so that the precise character of the pain can be identified. The patient will usually need help from the doctor in describing the pain by suggesting similarities or comparisons, such as hunger pain, burning pain, sticking pain. It is especially important that the presence or absence of cramping pain be established. The term *cramp* has many interpretations, so the patient will need to have an example of a cramplike pain that leaves no room for misinterpretation. One such example is to liken the pain to the wringing out of a washcloth with intense pain corresponding to wringing of the washcloth and abating when the washcloth is untwisted.

Abdominal pain can be characterized as cramping, dull, or aching and as either constant or intermittent.

Cramping abdominal pain is characterized by pain that increases in intensity in short waves to a maximum and then abruptly ceases for a period of complete absence of pain. Repetition of the pain occurs at intervals. This type of pain is associated with mechanical small bowel obstruction, and

when clearly present, it is pathognomonic of that condition. The interval between cramping pain helps locate the site of the obstruction with short pain-free intervals in more proximal obstructions and longer pain-free intervals in more distal obstruction.

Constant dull or aching abdominal pain is usually caused by distention or edema of the wall of a hollow viscus. Pain from stretch of the capsule of the liver and the spleen may also be perceived as dull or aching pain.

When a hollow viscus such as the gall bladder is distended, as is most common, from obstruction of the cystic duct, constant, dull, aching pain is experienced. When the gall bladder contracts against the obstruction, the stretch of the gall bladder wall is suddenly intensified, producing a crescendo of pain in addition to the constant dull ache already present (gall bladder colic).

Intensity

Each individual perceives pain intensity differently. The intensity of pain in an individual patient can often be assessed by the way in which the patient physically responds to it. Patients with peritonitis (somatic pain) invariably lie still, perhaps on one side or the other with the knees and hips flexed, whereas patients with pain arising from the hollow or solid viscera (visceral pain) do not lie still but constantly change position or move about.

Take, for example, a patient with the sudden onset of chemical peritonitis secondary to a perforated duodenal ulcer. The patient will immediately stop whatever activity he or she was doing at the moment of perforation, quickly sit or lie down, and remain as still as possible.

By contrast, a patient who has the onset of abdominal pain caused by acute distention of the gall bladder will not be able to find comfort by lying down in any position, but will move from chair to bed to the bathroom, where numerous attempts to relieve the pain by enemas, cathartics, self-induced vomiting, antacids, or other home remedies for "gas" pains are characteristic responses to visceral pain.

Localization

Visceral pain resulting from stretch of smooth muscle is localized in one of the three midline zones of the abdomen: epigastric, midabdominal, and lower abdominal. This midline zone pain, the sum of pain from the right and left splachnic pathways, is poorly localized, covers several body segments and, depending on its cause, varies from dull, aching, constant pain to cramping pain. Nausea, vomiting, pallor, and sweating are commonly associated with visceral pain.

Somatic pain is well localized. It is located asymmetrically and is intensified by jarring, deep inspiration or pressure on the abdominal wall.

One of the most useful physical examination procedures to identify the presence of abdominal pain of somatic origin is to ask the patient to distend his abdomen alternately by "pushing out" his umbilicus to touch the examiner's hand (held a few inches above the abdomen) and then to "suck in" his umbilicus to touch his spine. These subjective parietal peritoneum stretching procedures often identify the site of somatic pain by simple observation. It is especially useful in examination of children, as the subjective location of the pain without actual palpation of the abdomen by the physician may obviate misinterpretation from the child's anxiety. Since the cerebrospinal nerves that supply sensation to the anterior and lateral peritoneal surfaces are unilateral, any stimulation of the parietal peritoneal surfaces are lateralized quite well. Nausea, vomiting, pallor, and sweating are seldom associated with somatic pain.

It is well to have the patient indicate the site of the pain while standing and in the prone position to be certain of the true location.

Referred Pain and Accompanying Symptoms

Three cerebrospinal nerves, the phrenic, obturator and genitofemoral, are of particular importance because of the characteristic referred pain carried over these pathways in certain intra-abdominal conditions. Irritation, stretch, or injury of the dorsal or ventral aspects of the dome of the diaphragm produces referred pain in the supraclavicular fossa (Kehr's sign) corresponding to the sensory branches of the phrenic nerve (C3 through C5). Irritation of the genitofemoral nerve from such retroperitoneal inflammatory processes as retrocecal appendicitis or retroperitoneal perforation of the duodenum produces pain in the labia, testicle, or shaft of the penis on the involved side.

Irritation of the obturator nerve in the obturator fossa, usually from an incarcerated obturator hernia, produces pain along the medial aspect of the thigh to the knee (Howship–Romberg's sign).

Clearly, symptoms that accompany abdominal pain are important in making an accurate diagnosis. Among the most important are nausea, vomiting, abdominal distention, diarrhea, constipation, obstipation, tarry stools, chills, fever, urinary frequency, hematuria, and jaundice.

Basic Science

The splanchnic and the cerebrospinal are the two neural pathways available for transmission of abdominal pain. Pacinian corpuscles and free nerve endings in the walls of the viscera are the splanchnic afferent nerve receptors. They are sensitive only to stretch and spasm. By contrast, receptors of the cerebrospinal nerves are sensitive to pressure, friction, cutting, burning, and any other stimulus that can be appreciated by skin. In the dorsal root ganglia the splanchnic and cerebrospinal cell bodies are side by side. Their proximal fibers also terminate in close proximity within the spinal cord. The close relationship of these anatomic pathways may account for the fact that severe visceral pain, such as rapid distention of a viscus, may "spill over" into somatic segments (viscerosensory and visceromotor reflexes) in the absence of somatic nerve irritation. Understanding of "spillover" pain is essential for accurate diagnosis of abdominal pain.

Since the embryonic gut and its appendages arise as midline organs, their splanchnic innervation is bilateral, and accordingly, visceral pain is perceived in the midline. Cerebrospinal nerves to the parietal peritoneum (T6 through T12) have the same segmental arrangement as the lower thoracic dermatomes. There are no nerve fibers in the visceral peritoneum.

Clinical Significance

There is no substitute for a careful history of the type, onset, location, and progression of abdominal pain, as these symp-

toms closely match the pathogenesis of each disease process within the abdomen. This, coupled with understanding of the splanchnic and cerebrospinal innervation of the abdominal viscera, is essential for arriving at an accurate diagnosis in patients presenting with abdominal pain.

Differences in the location and rate of progression of lesions within the abdominal cavity may be summarized as outlined by Smith (1961) in terms of five possible components.

1. Visceral pain alone is a symmetric pain located in the midline anteriorly, with or without associated vasomotor phenomena.
2. On occasion, when visceral pain is of rapid onset and of great severity, at the peak intensity of the pain it may "spill over" at the spinal cord level by viscerosensory and visceromotor reflexes into the corresponding cerebrospinal pathways, producing somatic findings without pathologic involvement of somatic receptors.
3. Visceral and somatic pain often become combined as the causative lesion progresses from the viscus to involve adjacent somatic nerves. Visceral pain may continue, but a new and different pain is added.
4. Somatic pain may be so severe that it overshadows the visceral pain of origin in the affected viscus, making an accurate diagnosis difficult.
5. Referred pain due to irritation of the phrenic, obturator, and genitofemoral nerves are unique and diagnostically important findings remote from the abdomen that may provide clues to the source of abdominal pain.

The clinical significance of the pathways and stimuli responsible for the production of abdominal pain can perhaps best be appreciated by an analysis of the pathogenesis of *acute appendicitis,* as that disease process correlates with symptoms and physical findings common to that disorder.

The initiating event that starts the pathogenesis of the most common type of appendicitis is obstruction of the lumen of the appendix by a calcified concretion of fecal material called a *fecolith.* With obstruction of the appendiceal lumen, the continued production of large amounts of mucus from the rich concentration of goblet cells in the crypts of Lieberkuhn distends the appendix. At this stage in the pathogenesis of appendicitis, the pathologic diagnosis is an acute appendiceal mucocoele. Since stretch and distention are the only stimuli appreciated by the splanchnic (visceral) nerves, the characteristic midline, upper abdominal visceral pain of distention of a hollow viscus of embryologic origin from the midline, the classic periumbilical pain of early appendicitis begins. Although the appendix may be located almost anywhere in the abdominal or pelvic cavity due to its variable length as well as to rotation of the midgut, it is important to recognize that the epigastric or periumbilical visceral pain produced by distention will always be in the same midline upper abdominal location.

Since peristalsis in the appendix is absent, or at best ineffective, as demonstrated by the presence of a fecolith that otherwise would be extruded from the lumen, the visceral pain of distention of the appendix is characteristically a steady, dull, aching pain without intermittent waves of intensity.

It is of great value in the diagnosis of abdominal pain to attempt to establish both a pathologic and an anatomic diagnosis. For example, the pathologic diagnosis of "acute appendicitis" is not nearly as meaningful as a pathologic and anatomic diagnosis such as "acute suppurative right iliac fossa appendicitis." Understanding of the pain pathways and the types of stimuli responsible for initiating pain responses from the abdomen make precise pathologic and anatomic diagnoses possible.

The upper abdominal visceral pain of early appendicitis continues to increase gradually in intensity as the steady mucous production causes further distention of the obstructed appendiceal lumen. Intraluminal pressure progresses until the veins in the submucosa of the appendix become occluded by entrapment between the mucous membrane and the rigid, unyielding lamina propria in the appendiceal wall. At this stage in the pathogenesis, edema of the wall secondary to venous outflow occlusion rapidly ensues. This swelling, in addition to collection of edema fluid, causes a rather rapid, sharp increase in stretch of the appendiceal wall. This results in a marked increase in the intensity of the visceral pain. This point in the progression of the disease often prompts the patient to seek medical aid. The pathologic stage at the time of maximal swelling of the wall changes from an acute mucocoele to what is called *acute catarrhal appendicitis.* Since there is no inflammatory process in the appendix at this time, it is easily understood why there is no elevation of the white blood cell count or increase in body temperature.

As the pressures within the lumen and in the wall of the appendix continue to increase, interference with arteriolar blood flow in the submucosa ensues. At this stage the cells in the wall of the appendix, along with the mucosal lining of the lumen, begin to die. The stage is now set for invasion of the wall of the appendix by organisms from the lumen and the onset of acute suppurative appendicitis begins.

Obviously, as the wall of the appendix with the stretch-sensitive pacinian corpuscles dies, the dull, aching, intense upper midabdominal pain transmitted from these nerve endings disappears.

Further pain patterns of appendicitis now depend on its anatomic location. It has been said that there is no characteristic symptomatology of acute appendicitis. Not so: The symptoms of appendicitis are characteristic for each stage in the progression of the pathologic process and for each of the anatomic sites in which the appendix may be located.

The prodrome, produced by the early pathologic changes in the appendix and characterized by the visceral pain of distention, is the same no matter where the appendix is located, but the symptoms of suppuration and the continuing pathologic progression leading to gangrene, rupture, abscess formation, and local or generalized peritonitis differ markedly depending on the anatomic location of the appendix.

The appendix, as part of the midgut that rotates outside the abdominal cavity during embryologic development, may be located in at least nine locations: right iliac fossa, retrocecal, paraileal, retroileal, interloop, pelvic, right upper quadrant, left upper quadrant, and left iliac fossa.

The most common location of the appendix is in the right iliac fossa. In this location the inflammatory process in the suppurating appendix involves the contiguous parietal peritoneum. It is then that a new pain, entirely different from the visceral pain of the early stages and mediated over an entirely different neural pathway, begins. It is the sharp, well-localized, somatic pain of irritation of the parietal peritoneum transmitted to the dorsal root via the intercostal nerves. The patient points with one finger to the location of the pain, and jarring or any other stretch (deep breath, production of direct or indirect rebound tenderness) increases the local intensity of the pain.

Additional pathognomonic features of acute appendicitis in the right iliac fossa include straightforward motor and sensory reflex arcs that produce involuntary guarding over the involved irritated dermatome, as well as hyperesthesia similarly distributed.

Although it is often stated that the periumbilical pain in appendicitis "shifts" to the right lower quadrant, it is instructive to emphasize that the right lower quadrant pain is a new and entirely different pain. It is initiated by irritation of somatic nerve endings, transmitted by different neural pathways, and produces sharply localized symptoms and findings, in marked contrast to diffuse nature of visceral pain.

Examination of the symptoms and findings of acute suppurative appendicitis in a retrocecal location, another common position, serves to emphasize further the importance of an understanding of not only the anatomy and innervation of the peritoneal cavity but of the retroperitoneum and pelvis as well.

As has been stated, the visceral peritoneum has no somatic innervation. When the appendix is located retrocecally, for example, it is essential to remember that in this location it is retroperitoneal and, accordingly, has no contiguity with either visceral or parietal peritoneal serosa.

The early abdominal pain produced by stretch is present in its characteristic upper abdominal location, but as suppuration begins, there is no inflammatory involvement of parietal peritoneal surfaces, as in iliac fossa appendicitis, and consequently there is no localized right lower quadrant pain. The reason for absence of somatic pain when the appendix is retrocecal is obvious.

As the inflammatory process of retrocecal appendicitis continues, it may involve the psoas muscle, the obturator muscles, the ureter, and the genitofemoral nerve. Irritation of these structures is responsible for producing a positive psoas or obturator sign, white blood cells in the urine, and referred pain in the distribution of the branches of the genitofemoral nerve. The latter is manifested by pain in the testicle, shaft of the penis, or the labia on the right.

When the appendix is located in the pelvis, it must be remembered that the pelvis is not part of the abdominal cavity and that the pelvic parietal peritoneum receives its somatic innervation from the lumbosacral rather than the intercostal nerves. Accordingly, irritation of the pelvic parietal peritoneum is not recognized by the patient in a localized abdominal wall distribution.

It is helpful to recall that irritation of the pelvic parietal peritoneum usually produces localized pain in the midline suprapubicly, regardless of the location of the inflammatory process.

The preceding detailed discussion of some of the characteristic pain patterns seen in appendicitis and the pathology responsible for them serves to illustrate the diagnostic importance of accurate interpretation of abdominal pain based on anatomy and pathology.

Other Structures That Can Cause Pain

Upper abdominal organs have anatomic features that make pain patterns emanating from them far more complex than those of the appendix. Painful lesions of the gastroesophageal junction, the fundus and lesser curvature of the stomach, the biliary tract, and proximal portions of the duodenum commonly produce pain in the interscapular zone corresponding to the sixth thoracic segment, since the somatic innervation of the lesser omentum is supplied by that thoracic nerve. Pancreatic pain is often perceived in the same location one segment lower.

The stomach is so situated that portions of its surface are in contact with the diaphragm, the gastrohepatic ligament, the lesser sac, the pancreas, the parietal peritoneum, the splenic hilus, the gastrocolic ligament, the transverse mesocolon, and the transverse colon. *Inflammatory or neoplastic lesions of the stomach* that involve any of these surfaces may irritate somatic nerves from several different spinal segments. Accordingly, pain may be localized by the patient to the supraclavicular fossa from phrenic nerve stimuli, the interscapular region from irritation of T6 through T8, or even the lumbar region from involvement of the T12 through L1 spinal cord segments.

Similarly to the stomach, the duodenum is in anatomic relationship to a number of somatic cerebrospinal nerve roots. As a result, *perforating ulcer* pain can be appreciated in the interscapular zone, the right subcostal region, and the right lower quadrant depending on which somatic nerves are involved in the pathologic process. Retroperitoneal *perforation of the duodenum* from blunt abdominal trauma may cause irritation of the genitofemoral nerve from leaking duodenal contents resulting in pain in the right testicle or labia.

Pain from the gall bladder and biliary tract may have bilateral localization because they arise from outbudding of the midline gut and have bilateral splanchnic innervation. If the inflammatory process of *acute suppurative cholecystitis* involves the parietal peritoneum of the right upper quadrant, somatic pain with its usual local manifestations and referred pain along the involved cerebrospinal nerve to the tip of the scapula (T8) may be present. Involvement of the parietal peritoneum in the right upper quadrant from *suppuration of the gall bladder* is not a very common event, as the greater omentum (which has no somatic sensory innervation) often surrounds the inflamed gall bladder as a buffer between the inflammatory process and the parietes.

Pathologic conditions that arise from the *pancreas* are responsible for a broad spectrum of pain-producing syndromes. In addition, extrinsic lesions (e.g., penetrating duodenal ulcer) are frequently involved in the production of pain from the pancreas. Further, disruption of the integrity of the gland by pancreatitis permits the extravasation of enzymes that spread to many different intra-abdominal locations that may involve somatic spinal pathways from the phrenic nerve to the lumbosacral plexus.

The *small intestine,* like the rest of the midgut, produces upper midline, periumbilical visceral pain in response to distention or stretch. The foregut and hindgut are far less sensitive to stretch or distention. Pain from these portions of the intestinal tract, the stomach and duodenum from the former and the descending colon and rectum from the latter, is more often initiated by inflammatory lesions than from distention.

Experimental studies of the production of pain from the gastrointestinal tract in humans by inflation of balloons at various locations within the lumen of the gut should be interpreted very cautiously, as they bear little or no resemblance to actual pathologic conditions that produce pain in humans. The most valid observations concerning the origins of abdominal pain come from surgeons who have the advantage of prompt inspection of the site of pathology within the abdomen and the opportunity to compare these findings on the spot with the patient's perception of the abdominal pain.

The skin, subcutaneous tissues, fascia, muscle, and parietal peritoneum of the *abdominal wall* are richly supplied with somatic nerves from T6 through T12. Pain in the abdominal wall can result from neuromas in scars from previous laparotomies, such medical conditions as acute porphyria, or from herpes zoster. In addition, pain from trauma to the abdominal wall from blunt injury must be carefully identified to rule out abdominal pain originating from an intraperitoneal injury.

The ureters are second only to the pancreas as a source of abdominal pain caused by structures in the *retroperitoneum.* The renal pelvis is sensitive to distention, and the ureters are richly supplied with nerves from T10 through T12. Ureteral pain is ipsilateral, severe, and cramping in nature (renal colic). It is usually of such severity and located in the flank that the diagnosis is difficult to confuse with other abdominal catastrophies. Pain in the testicle or labia (T10) may on occasion confuse the diagnosis of renal colic with retrocecal appendicitis. The presence of red blood cells in the urinalysis may help solve this diagnostic dilemma.

Since the lower intercostal nerves also provide the parietal pleura and periphery of the diaphragm with sensory (somatic) innervation, as well as the abdominal wall and anterior peritoneal parietes, it is understandable that inflammatory processes that involve the *parietal pleura* innervated by these nerves may also be manifested by abdominal pain. Needless to say, an appendectomy is poor therapy for right lower lobar pneumonia that has produced reflex abdominal wall pain in the right lower quadrant.

Pericarditis, myocardial infarction, and pulmonary infarction may also cause inflammatory lesions that involve the *parietal diaphragmatic or thoracic pleura,* producing referred abdominal pain that may be misdiagnosed as a primary intraperitoneal disorder.

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